Original Article
EPIDEMIOLOGY/GENETICS

Obesity

Childhood Obesity and Device-Measured Sedentary Behavior: An Instrumental Variable Analysis of 3,864 Mother–Offspring Pairs

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Objective: Intergenerational data on mother–offspring pairs were utilized in an instrumental variable analysis to examine the longitudinal association between BMI and sedentary behavior.

Methods: The sample included 3,864 mother–offspring pairs from the 1970 British Cohort Study. Height and weight were recorded in mothers (age 31 [5.4] years) and offspring (age 10 years) and repeated in offspring during adulthood. Offspring provided objective data on sedentary behavior (7-day thigh-worn activPAL) in adulthood at age 46 to 47 years.

Results: Maternal BMI, the instrumental variable, was associated with offspring BMI at age 10 (change per kg/m^2, β = 0.11; 95% CI: 0.09 to 0.12), satisfying a key assumption of instrumental variable analyses. Offspring (change per kg/m^2, β = 0.010; 95% CI: −0.02 to 0.03 h/d) and maternal BMI (β = 0.017; 95% CI: 0.001 to 0.03 h/d) was related to offspring sedentary time, suggestive of a causal impact of BMI on sedentary behavior (two-stage least squares analysis, β = 0.18 [SE 0.08], *P* = 0.015). For moderate-vigorous physical activity, there were associations with offspring BMI (β = −0.010; 95% CI: −0.017 to −0.004) and maternal BMI (β = −0.007; 95% CI: −0.010 to −0.003), with evidence for causality (two-stage least squares analysis, β = −0.060 [SE 0.02], *P* = 0.001).

Conclusions: There is strong evidence for a causal pathway linking childhood obesity to greater sedentary behavior.

Study importance

What is already known?

- The association between sedentary behavior and obesity remains unclear.
- Methodological constraints make it difficult to shed light on causality and directionality.

What does this study add?

- We used an instrumental variable (maternal BMI) to examine associations between obesity and sitting.
- Our data support a causal pathway linking childhood obesity to greater sedentary behavior in adulthood.

How might the results change the direction of research or the focus of clinical practice?

- Policies to promote physical activity should focus on preventing childhood obesity and weight gain.

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Introduction

Popular media and scientific literature alike often portray sedentary behavior as a stand-alone risk factor contributing to the obesity epidemic. There is, however, little compelling evidence to support these claims (1,2). Evidence from observational study settings has often relied on cross-sectional designs and self-reported data on sedentary behavior (3). These methodological constraints make it difficult to shed light on causality and understand the direction of the association. Contemporary data have, in fact, suggested that the association more strongly operates in the direction from obesity to sedentary behavior/moderate-vigorous intensity physical activity (MVPA) rather than inactivity causing obesity (4-8). Nevertheless, existing studies have generally had short follow-up periods, and residual confounding remains a concern, particularly factors such as dietary intake. In addition, these studies were conducted on adult populations but have not examined associations across the life course from childhood to adulthood. From a policy perspective, it is important to understand whether early-life obesity drives sedentary

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behavior in adulthood, as this further highlights the importance of controlling childhood obesity for preventing poor behaviors that are likely to impact health outcomes in later life.

Instrumental variables have been used in various settings to overcome residual confounding. For example, Mendelian randomization utilizes genetic variants as an unconfounded instrument variable for the exposure of interest (9). This technique uses the rationale that genotypes are randomly allocated at conception, therefore associations of genetic variants with outcomes should be free of confounding and not influenced by reverse causation. In the absence of genetic data, an alternative instrumental variable analysis may be considered that utilizes parent–offspring data (10); it is well established that parental BMI is related to offspring BMI (11), and maternal–offspring relationships are likely to be less biased and confounded than conventional cross-sectional analyses using exposure and outcome data from the same individual. Conventional analyses would be unable to differentiate whether BMI drives sedentary behavior or more sedentary behavior leads to an increased BMI. In an instrumental variable analysis using maternal BMI as a proxy for offspring BMI, offspring sedentary behavior is unlikely to affect maternal BMI, thus mitigating problems of observational analyses and providing clarity on the direction of causality. Nevertheless, the parent–offspring instrument may still be confounded by shared intergenerational factors such as socioeconomic position.

The present study, therefore, aimed to assess causal associations between obesity in childhood and sitting behavior in middle age.

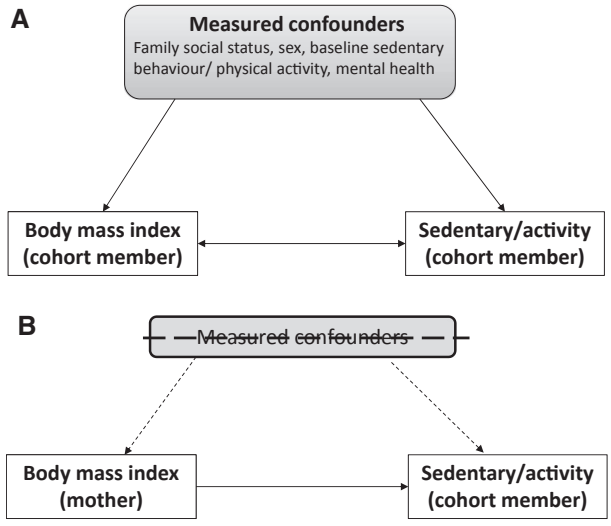

Figure 1 Hypothesized causal influence of BMI on sedentary behavior and physical activity. (**A**) In observational epidemiology, the association between the exposure (here, cohort member BMI) and the outcome of interest (here, cohort member sedentary behavior/physical activity) may be distorted because of confounding bias, and reverse causation. (**B**) Using maternal BMI as an instrument for cohort member BMI (where maternal BMI explains ~4% of the variance in offspring BMI) reduces the possibility of such limitations.

Methods

Design and participants

The 1970 British Cohort Study recruited participants born in a single week of 1970 from England, Scotland, and Wales (12). Participants have since been followed up on multiple occasions across their life. The present analyses incorporated data from the age-10-year and age-46-year surveys (Figure 1). The age-10 survey (1980) incorporated a medical assessment and contained questions on health-related behaviors completed by the mother of the cohort member. The age-46 survey was a home visit conducted in 2016/2017 and comprised a 60-minute face-to-face computer-assisted personal interview with biomedical assessments performed by trained nurses (13). The procedures followed were in accordance with the ethical standards of our institution/regional committee on human experimentation. Participants provided informed consent and received full ethical approval from various review boards across the different phases of data collection. The most recently conducted biomedical survey received ethics from the National Research Ethics Service Committee South East Coast–Brighton and Sussex (Reference Number 15/LO/1446).

BMI

At age 10 years and 46 years, height and weight of the cohort member were measured using standard protocols. Parents of cohort members self-reported their height and weight at the age-10 survey. BMI was calculated using the standard formula (weight in kilograms/height in meters squared, kg/m^2).

Sedentary behavior and activity

The study used the thigh-mounted activPAL monitor (activPAL3 micro; PAL Technologies Ltd., Glasgow, UK) as previously described (14). The device uses derived information about thigh position and acceleration to estimate body posture (i.e., sitting/lying and upright) and transition between these postures, stepping, and stepping speed (cadence). We employed a wear protocol previously designed to optimize high compliance (15); devices were programmed to sample at the default frequency of 20 Hz. The device was waterproofed and fitted by a trained nurse on the midline anterior aspect of the upper thigh as recommended by the manufacturer. Participants were requested to wear the device continuously for 7 days, including sleeping, bathing, swimming, and all physical activities, and were instructed not to reattach the device if it fell off or was removed before the stated end date. Devices were returned via mail at the end of the wear period. Data were processed using a previously validated (16) freely available software tool. The software uses an algorithm to isolate valid waking wear data from sleep or prolonged non-wear, summarized elsewhere (16). We used a step cadence threshold ≥ 100 in order to derive MVPA (17). The first partial day was removed, and subsequent days were defined from midnight to midnight. Participants were included if they recorded at least 10 hours of valid wear time on at least 1 day during the monitoring period.

Lifestyle and health measures

The cohort member's mother provided information regarding how often their child at age 10 watched TV and played sports (categorized as: never/sometimes/often) and completed questions from the Malaise inventory (18) to assess mental health of their child. Parents provided information on their occupation, which was categorized using the Office of Population Censuses and Surveys' 1970 and 1980 Classification of Occupations (managerial/professional/intermediate [skilled and nonskilled]/routine and manual).

Statistical analysis

Given the stronger intergenerational association between maternal BMI and offspring BMI compared with that derived from fathers and

offspring (19,20), the main analysis only used maternal BMI as an instrument for offspring BMI (Figure 1). We examined the distribution of offspring lifestyle and health variables according to maternal BMI, and effect estimates are presented as odds ratio and 95% confidence interval (CI) for a unit change (per kg/m^2) in maternal BMI. We compared results from the instrumental-variable estimates of the association between BMI and sitting time with results from standard linear regression. We performed an instrumental-variables regression analysis using the two-stage least squares regression "2SLS" command in SPSS to examine whether there was a causal impact of offspring BMI at age 10 on later-life sitting behavior and activity. Standard linear regression models were adjusted for sex, waking hours' activPAL wear time, and family social status (from father's social occupational group). We also utilized the longitudinal data to examine associations between change in BMI of the cohort member from age 10 years to age 46 years with sitting and activity levels at age 46 years. All analyses were conducted using SPSS software version 22 (IBM Corp., Armonk, New York).

Results

The flow of participants into the study is shown in Figure 2. At the age-46 survey, 88% of cohort members consented to wearing an ActivPAL device, and useable data were retrieved in 83% of the devices fitted. The analytic sample comprised 3,864 mother–offspring pairs. There were small differences between mothers of offspring included and excluded from these analyses. For example, mothers excluded were more likely to come from lower social occupational classes (39.8% vs. 36.1%, $P=0.001$) compared with the analytic sample, although there were no differences in age of mothers. The characteristics of the cohort members (offspring) in childhood and adulthood are presented in Table 1. During the wearable monitoring period, 90.7% of the sample recorded at least 3 full days of activPAL wear, and 65.5% wore the device for the full 7 days.

In order to establish the validity of maternal BMI as an instrumental variable, we examined associations with offspring BMI and potential confounding factors (Table 2). Maternal BMI was associated with offspring BMI at age 10 (change per kg/m^2, $\beta=0.11$; 95% CI: 0.09-0.12), satisfying a key assumption of instrumental variable analyses. However, several associations with potential confounders were also evident, including offspring TV viewing at age 10, severe behavioral problems, and family social status.

Scatterplots of the variables in our models are shown in the online Supporting Information. The effect estimates for associations between BMI (per kg/m^2) and sedentary time in offspring's adulthood were comparable when using offspring BMI at age 10 ($\beta=0.010$; 95% CI: –0.02 to 0.03 h/d) and maternal (as an instrument) BMI ($\beta=0.017$; 95% CI: 0.001 to 0.03 h/d) (Table 3). Instrumental variable analysis confirmed the likely causal impact of offspring BMI on sedentary behavior (two-stage least squares analysis, $\beta=0.18$ [SE 0.08], $P=0.015$). Similarly, for offspring MVPA in adulthood, there were associations with offspring BMI at age 10 (change per kg/m^2, $\beta=-0.010$; 95% CI: –0.017 to –0.004 h/d) and maternal BMI (change per kg/m^2, $\beta=-0.007$; 95% CI: –0.010 to –0.003 h/d), and strong evidence of causality was confirmed (two-stage least squares analysis, $\beta=-0.060$ [SE 0.02], $P=0.001$). We repeated the analyses with paternal BMI. Despite seeing an association between paternal and offspring BMI ($r=0.19$, $P<0.001$), there were no associations of paternal BMI with either offspring sitting time (change per kg/m^2, $\beta=0.005$; 95% CI: –0.01 to 0.02, $P=0.58$) or MVPA (change per kg/m^2, $\beta=-0.003$; 95%

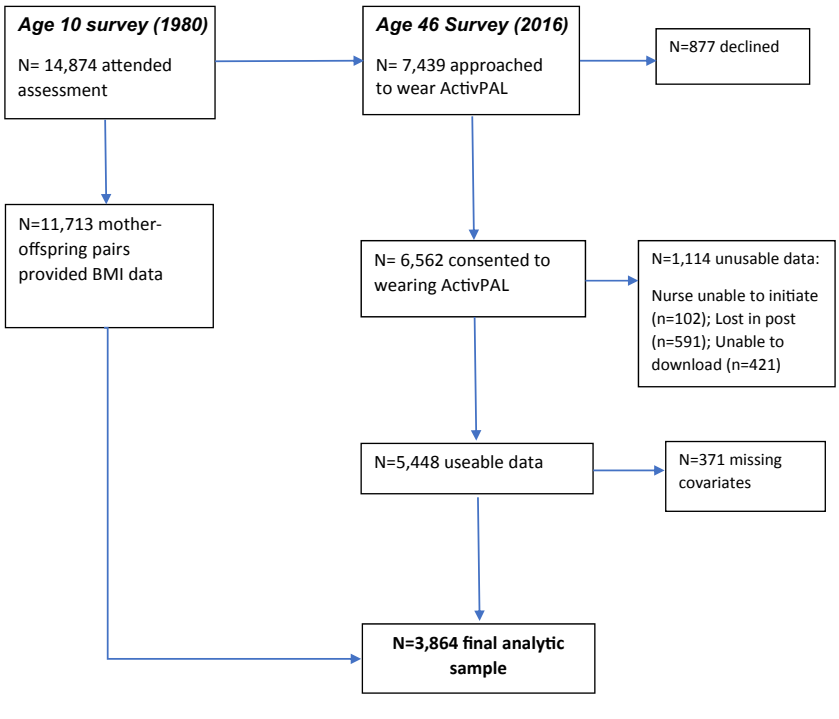

Figure 2 Flow of participants into study.

TABLE 1 Characteristics of cohort members ($N = 3,864$)

Age 10 years	
Sex (% male)	47.6
BMI (kg/m^2)	16.8 ± 2.0
TV viewing (% often)	79.1
Sports participation (% often)	53.1
Father's social occupational class (%)	
Professional	7.7
Managerial	28.6
Intermediate (skilled/nonskilled)	50.5
Manual/routine	13.2
Age 46/47 years	
BMI (kg/m^2)	28.1 ± 5.2
Sedentary time (h/d)	9.2 ± 2.0
Moderate-vigorous physical activity (min/d)	51 ± 25
Waking wear time (h/d)	15.8 ± 1.3
Average wear period (d)	6.2 ± 1.6
Education (% degree educated)	24.7
Smokers (%)	16.8
Self-rated health (% fair or poor)	15.0
Disability (% severely hampered, EU-SILC classification)	4.3

Data given as mean ± SD or %.
EU-SILC, European Union Statistics on Income and Living Conditions.

CI: −0.007 to 0.01, $P = 0.19$). In view of the findings showing associations between our instrumental variable and possible confounders, we also adjusted two-stage least squares analyses for offspring TV viewing (at age 10), sports participation, and malaise score, although this did not influence the associations.

We further explored associations between weight gain and sitting behavior/activity (Table 4). Participants with the greatest BMI increases from childhood to adulthood recorded greater sitting time and lower MVPA in adulthood after adjustment for a range of factors in both childhood and adulthood.

Discussion

We examined obesity and sedentary behavior across the life course using an instrumental variable approach utilizing intergenerational data on mother–offspring pairs. Our study has some unique methodological advantages. First, we employed the gold standard objective assessment of sitting time to prevent biases generated from self-reports. Second, to test the hypothesis that obesity drives sedentary behavior, we used maternal BMI as an instrument for offspring BMI to examine whether it is associated with offspring's sitting behavior in adulthood. We found evidence to support a causal pathway linking BMI in early life with greater device-measured sitting behavior in adulthood. This finding is largely consistent with a growing number of observational studies (4-8) that have suggested adiposity to be a stronger predictor of future sedentary behavior and lower MVPA rather than the reverse (i.e., activity predicting obesity). Additionally, a Mendelian randomization study employing genetic risk scores for obesity showed causal links between adiposity and lower levels of objectively assessed MVPA in teenagers (21); animal data have also

TABLE 2 Characteristics of study members at age 10 years according to tertiles of maternal BMI

		Offspring characteristics at age 10			
Maternal BMI (tertile)	BMI, mean (95% CI)	TV viewing (often)	Sports participation (often)	Low family SES (intermediate/routine and manual)	Malaise[b] (depression) score, mean (95% CI)
Lowest	16.3 (16.2-16.4)	76.8	50.9	9.9	9.1 (8.7-9.5)
Middle	16.8 (16.7-16.9)	79.3	53.6	12.3	8.9 (8.4-9.3)
Highest	17.3 (17.2-17.4)	81.6	55.0	17.9	9.1 (8.6-9.5)
Effect estimate[a]	β = 0.11 (0.09-0.12)	OR = 1.02 (1.01-1.03)	OR = 0.99 (0.98-1.01)	OR = 1.09 (1.08-1.10)	OR = 1.04 (1.02-1.06)

Data presented as mean (95% CI) or percentage.
[a]Effect estimates are presented as beta coefficient (β) or OR with 95% CI for a unit change (kg/m^2) in maternal BMI.
[b]Malaise was categorized into a binary outcome as severe behavioral problems (>95th centile malaise).
OR, odds ratio; SES, socioeconomic status.

TABLE 3 Association of BMI (age 10) with device-measured sitting and physical activity in adulthood (age 46)

	Minimally adjusted[a] β (95% CI)	Fully adjusted[b] β (95% CI)	Instrumental variable analysis estimation[a,c] β (95% CI), *P* value
Total sitting time			
Offspring BMI at age 10 years	0.010 (–0.02 to 0.03)	0.010 (–0.02 to 0.03)	0.18 (0.17 to 0.19), *P* = 0.015
Offspring BMI at age 46 years	0.047 (0.035 to 0.058)	0.047 (0.035 to 0.058)	
Maternal BMI	0.017 (0.001 to 0.033)	0.024 (0.008 to 0.039)	
MVPA			
Offspring BMI at age 10 years	–0.010 (–0.017 to –0.004)	–0.010 (–0.017 to –0.004)	–0.060 (–0.05 to –0.07), *P* = 0.001
Offspring BMI at age 46 years	–0.018 (–0.020 to –0.016)	–0.018 (–0.020 to –0.015)	
Maternal BMI	–0.007 (–0.010 to –0.003)	–0.007 (–0.011 to –0.003)	

[a]Adjusted for waking wear time, sex, and family social class.
[b]Adjusted for waking wear time, sex, family social class, offspring TV viewing and sports participation, and offspring malaise score.
[c]To examine whether there was a causal impact of offspring BMI at age 10 on later-life sitting behavior and activity.
Coefficients represent per kg/m^2 change in BMI; sitting and physical activity reflect h/d.

TABLE 4 Association of gain in BMI between childhood and adulthood with device-measured sitting behavior and MVPA at age 46

	Model 1 β (95% CI)	Model 2 β (95% CI)
Sitting	0.25 (0.19 to 0.31)	0.24 (0.18 to 0.31)
MVPA	–0.10 (–0.11 to –0.08)	–0.08 (–0.09 to –0.06)

β coefficients reflect h/d of objectively assessed sedentary time and physical activity in relation to gain in BMI (per SD) from 10 to 46 years old. Average gain in BMI = 11.6 ± 5.5 kg/m^2. Model 1 adjusted for sex, childhood (baseline) BMI, father social occupational group, and waking device wear time. Model 2 further adjusted for adulthood factors, including smoking, self-rated health, disability, and education.

suggested that inactivity is a consequence rather than a cause of obesity (22).

The variable chosen for the present study, maternal BMI, met the majority of assumptions required for an instrumental variables analysis. First, maternal BMI was positively associated with offspring BMI. Second, the offspring's sedentary behavior in middle age (outcome) could not plausibly affect variation in maternal BMI measured during their offspring's childhood. Thus, our instrumental variable analysis was considerably more protected from reverse causality than conventional analysis. Finally, there must be no other plausible pathway linking the instrumental variable with the outcome of interest except through its association with the risk factor of interest. This assumption was only partly met, as maternal BMI was weakly associated with one marker of offspring sedentary behavior (TV viewing in childhood), which might mediate the associations with the outcome (sitting behavior in middle age). However, the maternal influence on these confounding factors is likely to be weaker than that of the offspring, and adjustment for childhood TV viewing did not influence the results.

Mothers reporting higher BMI were from more socially deprived families, which may have contributed to a set of environmental circumstances driving sedentary habit formation, although we attempted to control for this by adjusting for social status. Dietary habits are likely to be transmitted from mother to child at an early age although robust data on dietary intake were not collected until the midlife assessment (13). Nevertheless, we observed no association between dietary energy intake and sedentary behavior in midlife (data not shown). It is unclear why mothers', but not fathers', BMI predicted offspring sedentary behavior and physical activity. The BMI of parents may have been a surrogate marker of their own physical activity levels although evidence of the influence of parental activity levels on their offspring is mixed (23,24).

The use of intergenerational, longitudinal data to reduce confounding and bias is a major strength of this study. While this analysis likely overcomes reverse causation found in traditional cross-sectional analyses, it may still be confounded by shared intergenerational factors. However, when we adjusted for such confounders, the results remained largely unchanged. We utilized objective assessments of sedentary behavior using a gold standard postural allocation technique, with high compliance with the wear protocol. Robust, objective data on sedentary behavior and physical activity in childhood were lacking. Also, given the absence of an instrumental variable for sedentary behavior in this study, we were unable to test the reverse association between sedentary time and obesity. Our instrumental variable explained approximately 4% of the variance in phenotype, although this is comparable to Mendelian randomization studies in which BMI allelic score explained ~2.8% of the variance in standardized BMI (21). The obesogenic environment has changed over the life course of cohort members in the present study, which may have influenced our findings. Indeed, obesity-related genes appear more strongly associated with BMI in more obesogenic home environments (25). We used mothers' self-reported weight and height. Previous studies, however, have demonstrated the validity of using self-reported weight (26). Furthermore, errors in self-reported weight are often systematic instead of random, reflecting both rounding to the nearest point of heaping and a tendency to report weights closer to ideal weight (27). There is some debate regarding indexing height to the power of 2 in the calculation of childhood BMI. Recent data have suggested that the best power (in terms of creating an index uncorrelated with height) depends on sex, age, and birth-year cohort as the variation in (and correlation between) weight and height is not constant across these three variables (28). For example, the best power in the 1970 British Cohort Study at 10 years is around 2.4 in girls. The correlation between kg/m^2 and kg/m$^{2.4}$ is, however, extremely high ($r > 0.95$) and thus should not have influenced our results. Over 95% of the cohort was "British white," therefore it was not possible to investigate possible interactions by ethnicity, nor are the data generalizable to nonwhite populations.

In conclusion, to our knowledge this is the first study to adopt a novel approach utilizing intergenerational data on mother–offspring pairs to examine a causal pathway linking childhood BMI to sedentary behavior in adulthood. Our findings suggest that obesity in early life may be causally related to adverse sitting and physical activity behaviors in adulthood, potentially further amplifying the risks of obesity and other cardiometabolic conditions. Policies to promote physical activity should focus on preventing childhood obesity and weight gain.O

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Author contributions: MH had full access to the data and takes responsibility for the integrity and accuracy of the results. All authors contributed to the concept and design of study, drafting, and critical revision of the manuscript.

Data sharing statement: Full data are available at the UK data archive at http://www.data-archive.ac.uk/.

Supporting information: Additional Supporting Information may be found in the online version of this article.

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